greatest over the carboxyl-terminal two thirds of the three proteins. The CKB3 protein shares most of the structural features of CKB1 and CKB2 at the level of primary structure (10). First, CKB3 contains a potential metal-binding motif Cys-Pro-$X_3$-Cys-$X_{22}$-Cys-Pro-X-Cys (SEQ I.D. No. 3) (45) (wherein "X" may be any amino acid). Second, although the conserved autophosphorylation site, Ser-Ser-Ser-Glu-Glu (SEQ I.D. No. 4), is missing in the amino-terminal region of CKB3, there are two CK2 recognition phosphorylation sites, $^{81}$Ser-Gly-Ser-Glu-Gly-Asp (SEQ I.D. No. 5) and $^{83}$Ser-Glu-Gly-Asp-Asp, (SEQ I.D. No. 6) in about the same location as in the animal β-subunits. Third, CKB3 has an N-terminal extension preceding the putative phosphorylation sites which exhibits a moderate level of similarity to the N-terminal extension of the other *Arabidopsis* β-subunits. Neither yeast nor animal β-subunits contain such an N-terminal extension, and this region bears no extensive similarity to other proteins.

Please replace the three paragraphs starting at line 10, page 19, with the following text:

**Overexpression of *CKB3*** To further explore the hypothesis that the CK2-CCA1 DNA-protein complex plays a role in the regulation of the circadian clock, we created transgenic *Arabidopsis* plants overexpressing a c-myc tagged form of *CKB3* and analyzed their circadian behavior. To produce the tagged *CKB3 an* Eco RI-Bsr GI fragment of the plasmid pUC-CKB3 that contains the entire coding sequence of *CKB3* cDNA at the Bam HI site of pUC19 was replaced with the duplex DNA composed of oligonucleotide myc-CKB3F (5'- AATTGAGATCTCATGGAGCAAAAGCTTATCAGCGAGGAGGACTT GAACAT) (SEQ I.D. No. 7) and oligo-nucleotide myc-CKB3B (5'- GTACATGTTCAAGTCCTCCTC GCTGATAAGCTTTTGCTCCATGAGATCT) (SEQ I.D. No. 8) to introduce the Bgl II site and c-myc encoding sequence in front of *CKB3*. The resultant plasmid was digested with Bgl II and Hinc II, and the Bgl II-Hinc II fragment was subcloned into the pBI121 vector (Clonetech). This construct was used to transform *Agrobacterium tumefaciens* strain A2260, and then *Arabidopsis* plants

(Columbia ecotype) using the *in planta* transformation procedure as described (61, 62). Overexpression of *CKB3* had no apparent effects on plant growth and development except timing of flowering.

From 16 transgenic lines that each had a single site of insertion, two transgenic lines designated ox18 and ox41 were further analyzed. Levels of *CKB3* transcript in the fourth generation of homozygous *CKB3*-overexpressing (*CKB3*-ox) plants were approximately 20 times higher than that in the wild-type (Fig. 10A). Ten μg of total RNA were treated with RQ1 RNase-free DNase (Promega) and the first-strand cDNA was synthesized as described in (43) The product of the first-strand synthesis was then used for PCR to amplify 140 bp *CKB3* cDNA with the primers CKB3F1 (5'-ACAAGGAACGTAGTGGAGGAGGTG) (SEQ I.D. No. 9) and CKB3B3 (5'-AACCCTAGATGT GGTGGTGGAAG) (SEQ I.D. No. 10). As a control, primers UBQ10-5' and UBQ10-3' (61, 62) were used to amplify 111 bp *UBQ10* cDNA. The resultant PCR fragments were separated on a 2% agarose gel, blotted and hybridized with $^{32}$P-labeled probes.

The transgenic plants contained appreciable amounts of the c-myc-tagged CKB3 protein (Fig. 10B). Protein extracts were obtained by grinding 10-day-old seedlings in 100 μl of 3XSDS-sample buffer (180 mM Tris-HCl (pH 6.8), 6% SDS, 30% glycerol, 7.5% 2-mercaptoethanol), boiling this mixture for 5 min and saving the supernatant after centrifugation for 15 min at 14,000x*g*. Protein concentration was measured with a protein assay reagent (Bio-Rad). Western blots were performed using anti-c-myc monoclonal antibody 9E10 (17, 15, 64, 46) following the methods described in (61, 62).Measurement of CK2 activity in whole-cell extracts showed that the transgenic lines exhibited a 1.7-fold increase in CK2 activity (Fig. 10C). Frozen seedlings (100 mg) were ground and extracted with 100 μl of extraction buffer (50 mM Tris-HCl (pH 7.5), 15 mM $MgCl_2$, 0.1 M KCl, 0.25 M sucrose and 10% glycerol, 1 mM phenylmethylsulfonylfluoride, protease inhibitor cocktail (Boehringer Mannheim), phosphatase inhibitor mixture (55, 26, 48) and 14 mM 2-mercaptoethanol). After centrifugation at 14,000x*g* for 15 min, the supernatant

was saved and protein concentration was measured as above. CK2 assays were carried out at 37 °C with 200 μM CK2 specific peptide substrate Arg-Arg-Arg-Asp-Asp-Asp-Ser-Asp-Asp-Asp (SEQ I.D. No. 11) (Boehringer Mannheim) in 25 μl of CK2 buffer (55, 26, 48) as described (5).